

***Helicobacter* Species Strain Mainz Isolated from Cultures of Blood from Two Patients with AIDS**

Campylobacter species are known to cause enteric disease in immunocompetent and immunodeficient patients, and systemic illness (bacteremia) in immunocompromised patients such as those infected with HIV [1]. *Helicobacter cinaedi* and *Helicobacter fennelliae*, formerly *Campylobacter cinaedi* and *Campylobacter fennelliae* [2], are associated with symptomatic or asymptomatic bacteremia, proctocolitis, cellulitis, meningitis, and gastroenteritis [3]. The *Helicobacter* species strain Mainz described recently has been isolated from the knee of a patient with AIDS and septic arthritis [4]. We report bacteremia with *Helicobacter* species strain Mainz in two patients with AIDS who presented with fever.

Patient 1. A 29-year-old HIV-infected homosexual man with a history of cerebral toxoplasmosis (January 1994) was receiving antitoxoplasmic maintenance therapy with pyrimethamine and sulfadiazine. In July 1995, he presented with fever and cough. His CD4 lymphocyte count was $.03 \times 10^9/L$ and findings on his chest radiograph were normal. The FAN aerobic blood culture bottle (BacT/Alert; Organon Teknika, Turnhout, Belgium) revealed an elevated growth index after 2 days of incubation, without microscopically detectable microorganisms; subcultures became positive for gram-negative *Campylobacter*-like organisms, not further specified after 3 days of growth under microaerophilic conditions at 37°C. His condition improved after empirical treatment with clarithromycin for 10 days.

Four months later, in November 1995, the patient's condition deteriorated; he developed fever and diarrhea and was hospitalized. An aerobic blood culture and the subculture under microaerophilic conditions became positive for *Campylobacter*-like organisms, later identified as *Helicobacter* species strain Mainz. A stool culture was not performed. The patient was treated empirically with oral ciprofloxacin for 10 days. The fever and diarrhea abated, and the patient was released from the hospital. He died of a wasting syndrome at the end of January 1996.

Patient 2. A 33-year-old HIV-infected patient developed polymyositis in 1993 that required maintenance therapy with prednisone. In 1995, esophageal candidiasis was diagnosed and treated. In spring 1996, the patient presented with signs and symptoms of pancreatitis. His CD4 lymphocyte count was $.01 \times 10^9/L$. After he abstained from food and didanosine therapy was discontinued, there was initial improvement in the patient's condition, and serum amylase values decreased. Because of a 1-day episode of fever, two blood cultures were performed and yielded a *Campylobacter*-like organism, later identified as *Helicobacter* species strain Mainz. The patient received empirical treatment with ciprofloxacin for 10 days. Because the pancreatitis worsened and his condition again deteriorated, rehospitalization was necessary about 1 week later. None of the six blood cultures performed became positive at this time. The abdominal pain resolved after discontinuation of therapy

with co-trimoxazole, which the patient was receiving as chemoprophylaxis for *Pneumocystis carinii* pneumonia. The results of abdominal ultrasonography and endoscopic cholangiopancreatography were normal. No relapse of the bacteremia was observed during the 1-year follow-up period.

For both patients, the aerobic FAN blood cultures (BacT/Alert) showed a positive growth signal after 1–3 days of incubation, but conventional aerobic subcultures remained negative after 3 days. Microaerophilic subcultures on blood agar were performed, as suggested by Kiehlbauch et al. [3] for the isolation of *Helicobacter cinaedi* in immunocompromised hosts; small colonies of *Campylobacter*-like organisms grew after 3 days. The same curved, sometimes spiral gram-negative bacteria could also be detected by gram stain of the broth from the positive blood cultures.

The isolates were subjected to phenotypic testing and were identified by hybridization with genomic DNA of representative *Campylobacter* and *Helicobacter* species in a nonradioactive dot-blot procedure [5] as well as partial sequencing of 16S ribosomal RNA. Both results of conventional biochemical testing [6] and alignment of ribosomal RNA sequences concurred in placing the strains in the genus *Helicobacter*, the most closely related species being *H. cinaedi*, *H. fennelliae*, and *Helicobacter pullorum*. The overall DNA homology of the two isolates with labeled DNA from *Helicobacter* species strain Mainz was 83%; none of the named *Helicobacter* or *Campylobacter* species showed significant DNA homology under stringent conditions. The available evidence thus clearly shows that the two strains from the patients described above belonged to the same species as the single isolate of *Helicobacter* species described as "strain Mainz" in the literature [4].

Although evaluation of the febrile episodes did not reveal any other opportunistic infections in these two patients with AIDS, it remains unclear whether there was any relation between the clinical manifestations and the bacteremia due to *Helicobacter* species strain Mainz. The spectrum of illness caused by this organism remains to be defined. This report sustains the practice of performing microaerophilic subcultures on blood cultures with positive signals [3].

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Successful Treatment of Multicentric Castleman's Disease in a Patient with Human Immunodeficiency Virus Infection

Castleman's disease is an atypical lymphoproliferative disorder characterized by lymph node hyperplasia with germinal center formation and marked capillary proliferation. Multicentric Castleman's disease (MCD) has been described more recently, most frequently in patients infected with HIV [1]. An association between infection with human herpesvirus 8 (HHV-8) and MCD has now been demonstrated [2, 3], and HHV-8 has been detected in the lymph nodes and/or peripheral blood mononuclear cells of patients with MCD at various stages of HIV disease [4]. The clinical courses of patients with MCD have been altered by treatment with corticosteroids and various chemotherapeutic agents, but, overall, the prognosis has been poor. We report a sustained response to therapy in a patient with HIV infection and MCD.

A 29-year-old male with HIV disease, a CD4 cell count of 30/mm³, and no history of opportunistic infections was admitted to the hospital on 17 May 1996 because of fever, night sweats, and shortness of breath of 3 weeks' duration and a 12-kg weight loss during the prior 3 months. He had a WBC count of 1,100/mm³ with 50% neutrophils, a hematocrit of 21.9%, and a platelet count of 132,000/mm³. His temperature was 40.0°C. Physical examination revealed left anterior cervical adenopathy and an enlarged liver. Therapy with antibiotics and granulocyte colony-stimulating factor was started, without improvement in his condition. A CT scan of the abdomen and chest revealed bilateral hilar adenopathy, small bilateral pleural effusions, hepatosplenomegaly with multiple hypodense lesions in the liver and spleen, and retroperitoneal lymphadenopathy. Scintigraphy with gallium-67 showed no abnormal uptake. Findings on examination of a bone marrow biopsy specimen were normal, and culture of the specimen was sterile.

One month later he was readmitted with similar symptoms. He remained pancytopenic. Examination of a liver biopsy specimen revealed nonspecific reactive hepatitis. Splenic biopsy showed clusters of atypical spindle and lymphoid cells. An assay for tumor markers was not done because an insufficient quantity of tissue was obtained. He started receiving treatment with stavudine (40 mg twice daily), lamivudine (150 mg twice daily), and zalcitabine (600 mg twice daily).

In August 1996 the patient developed a persistent cough. Radiographs of the chest showed bilateral hilar adenopathy and perihilar infiltrates. A CT scan of the chest with contrast medium revealed a 2.2 × 3.0-cm mass in the azygoesophageal recess that encased the right middle-lobe bronchus, as well as a right middle-lobe infiltrate and an 8-mm left upper-lobe nodule. Diagnostic splenectomy and exploratory laparotomy were performed on 7 September 1996. Pathological review of the para-aortic lymph node and spleen revealed Castleman's disease. PCR analysis of splenic tissue was strongly positive for HHV-8 DNA. The patient began receiving foscarnet (3,600 mg every 8 hours) 2 days after surgery. His fever resolved within 24 hours after splenectomy, and his pulmonary symptoms resolved within 1 week. After 3 weeks, he continued to receive daily treatment with foscarnet (5,400 mg) for an additional 4 weeks. During this time he remained well and gained 14 kg. His pancytopenia resolved. A CT scan of the chest, obtained 2 months later, showed complete resolution of his pulmonary disease. After 12 months, he remains well, with a CD4 cell count of 342/mm³.

To our knowledge, a sustained response to therapy for MCD in a patient with HIV infection has not been described previously. Our patient underwent splenectomy and received antiretroviral therapy and antiviral therapy directed against herpes viruses. Splenectomy has been associated with improved survival [5] and resolution of some opportunistic infections [6] in patients with HIV disease. It is difficult to determine whether our patient recovered because of a specific intervention or a combination of antiviral therapies and splenectomy.

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